

EXHIBIT C-1

Brent C. Staggs, M.D.

Diplomat, American Board of Pathology

1 Lile Court, Suite 101

Little Rock, Arkansas 72205

Phone (501) 831-0161

Fax (501) 202-1420

brentstaggs@comcast.net

Original report: April 25, 2019

Supplemental report: June 19, 2019 (death certificate)

RE: Toy Sr., Thomas H. (DOB May 25, 1935)

Dear Sirs,

You have asked for my opinion regarding the diagnosis of Mr. Toy's disease, and whether his disease was caused by exposure to asbestos. I have reviewed medical records and pathology materials of Mr. Toy, as well as a work history containing asbestos exposure information provided by the Dean, Omar, Branham, Shirley law firm.

CLINICAL HISTORY:

Progress note (July 25, 2013) indicated evaluation of an abnormal chest x-ray with a history of asbestos related pleural disease, asbestosis and a history of smoking. Smoking history was reported as one pack per day for 30 years having quit in 1970. Prior work for the government and shipyards as a machinist reportedly resulted in significant asbestos exposure for about 14 years. An abnormal chest x-ray was evaluated. CT scan from May 2013 reportedly showed bilateral calcified pleural plaques and mild sub pleural fibrosis at the lung basis. Mr. Toy reportedly denied shortness of breath. Social history again indicated past occupational asbestos exposure. Physical examination identified bilateral crackles upon auscultation. The assessment indicated Mr. Toy had significant asbestos exposure during work and shipyards for about 14 years

with CT scan showing evidence of asbestos related pleural disease and the examination including CT scan confirming mild asbestosis.

Pulmonary consultation note (September 19, 2018) indicated a chief complaint of pleural effusion with a history of benign prostatic hypertrophy, chronic kidney disease, hypercholesterolemia, hypertension and asbestosis. Progressive shortness of breath was noted over the preceding two – four weeks. Prior CT scan reportedly showed bilateral calcified pleural plaques and a recent chest x-ray identified a moderate left pleural effusion and pleural plaques. The assessment indicated pleural effusion and pneumoconiosis due to asbestos.

Procedure note of thoracentesis (September 26, 2018) indicated obtaining 1760 mL of hemorrhagic pleural fluid.

Consultative report (September 27, 2018) indicated presentation with a left pleural effusion. CT scan identified bilateral pleural plaques consistent with the history of asbestos exposure. Social history indicated prior asbestos exposure while working for the government. A history of smoking one pack of cigarettes per day for 20 years, 40 years previous, was reported.

Operative report of video assisted thoracoscopy identified multiple areas of pleural plaque covering the entire pleura from the apex to the diaphragm within the left chest. Multiple biopsies were obtained.

Consultative report (October 4, 2018) indicated a diagnosis of “malignant mesothelioma”. A history of prior asbestos exposure was reported. Multiple pleural based nodules were reportedly biopsied and proven to be malignant mesothelioma. Malignant mesothelioma was deemed to be unresectable. Treatment options were discussed.

Discharge summary (October 4, 2018) indicated a primary diagnosis of “malignant mesothelioma”.

Office note (October 10, 2018) indicated presentation for discussion of treatment options regarding a new diagnosis of malignant pleural mesothelioma, epithelioid type.

Death certificate from the state of California listed the cause of death as “mesothelioma, etiology unknown”.

RADIOLOGY REPORTS:

Chest x-ray (September 15, 2018) identified right pleural calcification likely representing calcified pleural plaque.

CT scan (September 27, 2018) identified a small residual left pleural effusion and asbestos related pleural disease with probable parenchymal scarring in the right lung base.

PATHOLOGY REPORTS:

Report from Butler Memorial Hospital (CN 18 – 1509, September 27, 2018) diagnosed a left pleural fluid specimen as “atypical cells present”. Atypical cells reportedly sustained positive for 65/6, calretinin WT1 but negative for MOC 31, TTF1 and Napsin A.

Report from Butler Memorial Hospital (CN 18 – 1524, October 1, 2018) diagnosed a left pleural fluid specimen as “atypical cells present”.

Report from Butler Memorial Hospital (SU 18 – 16234, October 1, 2018) diagnosed multiple left pleura biopsies all as “malignant mesothelioma”. The comment reported tumor cells positive for calretinin, 65/6, WT1, D2 40, CD 141 and focal/weak MOC 31 but negative for TTF1 and Napsin A. The histologic and staining patterns are all reported as supporting the diagnosis of malignant mesothelioma.

PATHOLOGY MATERIALS:

I received one paraffin block label “CN 18 – 1509”. I created one H&E stained glass slide. Microscopic examination demonstrated cellblock section of a fluid specimen showing blood elements along with clusters of large malignant epithelioid cells. Scattered malignant cells showed large regular, round and oval nuclei with multiple prominent nucleoli and voluminous eosinophilic cytoplasm. Tumor cells were arranged in small clusters. No gland, mucin or keratin formation was detected.

I received one paraffin block labeled “CN 18 – 1524”. I created one H&E stained glass line. Microscopic examination demonstrated cellblock sections of a fluid specimen showing copious blood elements and innumerable clusters of large malignant epithelioid

cells. Tumor cells were identical to those described above showing large pleomorphic nuclei with vesicular chromatin and abundant cytoplasm.

I received three paraffin blocks all labeled "SU 18 – 16234". I created one H&E stained glass slide from each block. Microscopic examination demonstrated sections of parietal pleura and chest wall soft tissue extensively involved by a malignant epithelioid neoplasm. Tumor cells were again identical to those seen in fluid specimen. Tumor cells were large with moderate to marked pleomorphism showing larger regular come around and oval nuclei with vesicular chromatin, mitotic figures and abundant eosinophilic cytoplasm. Tumor cells were arranged in nests and sheets with destructive invasion throughout pleura and into chest wall soft tissues. No gland, mucin or keratin formation was detected. No alveolar lung parenchyma was available for review.

Based on my review of records and tissues, my diagnosis is as follows:

Pleural fluid, left, thoracentesis (CN18 – 1509 & 1524):

- **Primary Pleural Malignant Mesothelioma, epithelioid type.**

Parietal pleura, left, biopsies (SU 18 – 16234):

- **Primary Pleural Malignant Mesothelioma, epithelioid type.**

ASBESTOS EXPOSURE HISTORY:

An asbestos exposure history letter provided by the Dean, Omar, Branham, Shirley law firm reports Mr. Toy was exposed to asbestos from approximately 1953 until 1980 while serving in the United States Army, as a Marine machinist at Hunters Point Naples Shipyard, and as a maintenance machinist at Treasure Island Naval Station. From approximately 1953 through 1959, Mr. Toy served as a wheel vehicle mechanic and then a motor sergeant. His job was to maintain the Army's fleet of vehicles during this time. The Army had approximately 50 2 ½ ton, ¾ ton, and ¼ ton vehicles. Mr. Toy performed brake, clutch, and gasket work on the vehicles, including sending of new clutches and brakes and blowout of brake drums and clutch discs with compressed air during removal. He worked in a motor pool next to other mechanics. The motor pool had 50 bays. There was no ventilation. Spent eight hours a day during this time period in the

motor pool working on and around others working on the Army's fleet of vehicles. He swept up dust from his work every day.

From approximately 1962 through 1973, Mr. Toy worked as a Marine machinist at Hunter's Point Naval Shipyard. As a result, Mr. Toy was exposed daily to asbestos containing gaskets and insulation associated with valves, pumps, turbines, generators, compressors and steam traps. His duties included the installation and removal of asbestos-containing gasket materials and the removal of asbestos-containing insulation materials. From approximately 1974 until 1980, Mr. Toy worked as a maintenance machinist at Treasure Island Naval Shipyard. He received daily exposure from asbestos gaskets and insulation associated with steam traps, valves, pumps and compressors. His duties included daily removal and replacement of asbestos-containing insulation and gaskets associated with valves, pumps, and compressors, and the installation and removal of brakes and clutches as a professional vehicle mechanic for approximately six months.

Mr. Toy was also exposed to asbestos-containing brakes, clutches and gaskets while performing work on his family vehicles from the early 1950s until 2016.

SUMMARY:

Based on my review of the medical records including pathology materials, I conclude that Mr. Toy had malignant mesothelioma of the pleura. The exposure history of Mr. Toy provided to me identifies a significant history of asbestos exposure. Mr. Toy developed malignant mesothelioma after an appropriate latency period following his first known exposures to asbestos.

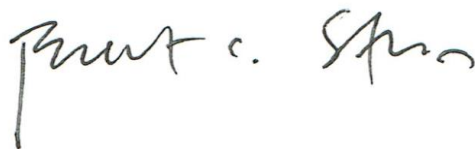
The occurrence of mesothelioma is associated with asbestos exposure, and mesothelioma is considered by many sources to be a signal tumor, meaning it signals prior asbestos exposure. Scientifically and medically speaking, it is not one asbestos fiber or one exposure, to the exclusion of other exposures that causes a person's mesothelioma. Mesothelioma is caused by the totality of asbestos exposures, often called cumulative dose, that an individual is exposed to over his or her lifetime, taking into account an appropriate latency period. A mesothelioma occurs only after repeated exposures to asbestos over time, which in turn causes repeated damage to the mesothelial DNA and leads to mutations. It is well established that even brief or low

exposures to asbestos can cause mesothelioma; however, it is also important to recognize that mesothelioma is a dose response disease. That means from an epidemiologic perspective, the greater the dose of asbestos, the greater the chance a given individual will develop mesothelioma, thus smaller exposures contribute smaller risks and larger exposures contribute larger risks. It is well established that exposure to both chrysotile and amphibole asbestos causes malignant mesothelioma. When I review an individual's exposure to asbestos and evaluate the causation of disease, as I have done in this case, I do not state that any contributor to the cumulative dose, no matter how small, is a significant factor to the development of mesothelioma. Rather, I review, evaluate, and consider the information available to me about an individual's identified exposures to asbestos, and only after that review will I consider causation and attribution of the asbestos exposures. My review is done in the context of additional opinions and foundation as stated in my affidavit of April 15, 2016, provided separately or attached to this report.

Based on the information available to me, that I have reviewed in this case and is described above, Mr. Toy had significant exposures to asbestos over his working lifetime. It is my opinion to a reasonable degree of medical certainty that Mr. Toy has a malignant mesothelioma that was caused by these identified and substantial exposures to asbestos.

I will supplement or amend this report as appropriate, if additional information becomes available.

Sincerely,

A handwritten signature in black ink, appearing to read "Brent C. Staggs". The signature is written in a cursive, flowing style with a large initial 'B'.

Brent C. Staggs, M.D.